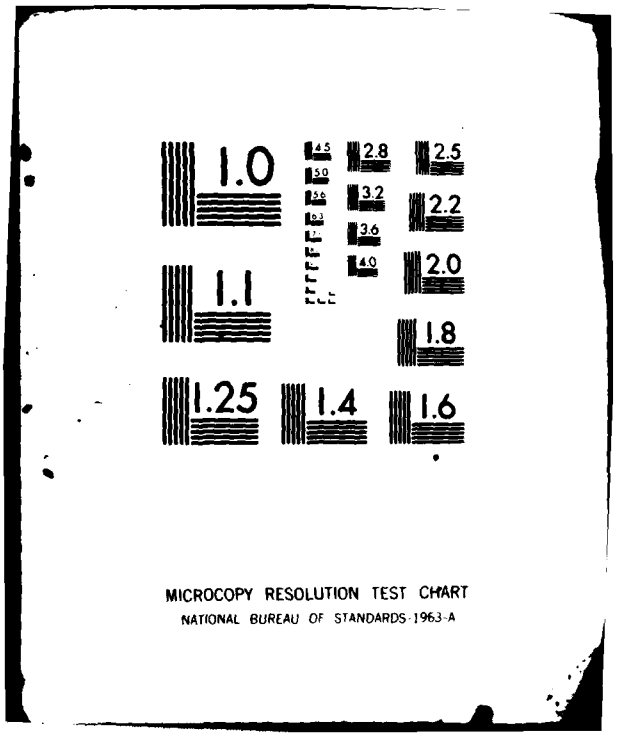
AD-A108 210 ARMY MEDICAL RESEARCH INST OF INFECTIOUS DISEASES FR--ETC F/G 6/5
EFFECT OF INFECTION ON NUTRIENT METABOLISM.(U)
SEP 81 R W WANNEMACHER

UNCLASSIFIED NL

END
DATE FILMED
-82
DTIC


1.0
1.1
1.25
1.4
1.6
1.8
2.0
2.2
2.5
2.8
3.2
3.6
4.0
4.5
5.0
5.6
6.3
MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS-1963-A

UNCLASSIFIED
SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER	2. GOVT ACCESSION NO. AD-A108 210 / 3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) Effect of Infection on Nutrient Metabolism	5. TYPE OF REPORT & PERIOD COVERED Interim
	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) Robert W. Wannemacher, Jr.	8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS U.S. Army Medical Research Institute of Infectious Diseases, SGRD-UIP-S Fort Detrick, Frederick, Maryland 21701	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 66102B 1M166102BS10-AQ-175
11. CONTROLLING OFFICE NAME AND ADDRESS U.S. Army Medical Research and Development Command, Office of the Surgeon General Department of the Army, Washington, DC 20314	12. REPORT DATE 17 Sep 81
	13. NUMBER OF PAGES 4 pages
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)	15. SECURITY CLASS. (of this report)
	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release - distribution unlimited

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

Reprints bearing assigned AD number will be forwarded upon receipt.
To be published in Nutrition and the M.D.

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Infect, protein, carbohydrate, lipid, nutrition.

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

A short review on effects of infection on nutrient requirements and metabolism.

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

UNCLASSIFIED 405039

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

Effect of Infection on Nutrient Metabolism

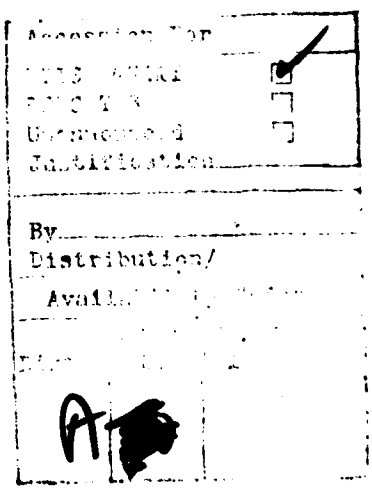
Robert W. Wannemacher, Jr., Ph.D.

Chief, Dept. of Pathophysiology and Therapeutics

Physical Sciences Division

U.S. Army Medical Research Institute of Infectious Diseases

Fort Detrick, Frederick, Maryland



Effect of Infection on Nutrient Metabolism

Robert W. Wannemacher, Jr., Ph.D.
Chief, Dept. of Pathophysiology and Therapeutics.
Physical Sciences Division.
U.S. Army Medical Research Institute of Infectious Diseases.
Fort Detrick, Frederick, Maryland.

Acute clinical illness caused by an infectious process is typically characterized by a catabolic response, which includes loss of body weight, wasting of body protein, anorexia, increased metabolic rate, and negative nitrogen, potassium, phosphorous, magnesium, sulfur, and zinc balances. Part of the catabolic response can be reversed in laboratory animal models by providing an adequate supply of amino acid and calories via oral or intravenous route.

Alterations in protein metabolism

During acute infectious illness in the presence of anorexia, that is usually associated with infectious disease, a continual loss of body nitrogen results in negative nitrogen balance. The severity of loss in nitrogen, however, cannot be accounted for by the anorexia alone. Rather, the severity and duration of fever during acute infections appear to be the major contributors to the protein wasting observed. Protein of skeletal muscle appears to be the major contributor to the increased loss of body protein and nitrogen during infectious disease. Amino acids derived from the breakdown of muscle protein are utilized at an increased rate as energy sources, gluconeogenic and ureagenic substrates, and for the synthesis of proteins associated with host defense mechanisms. While total body protein synthesis is decreased during the catabolic phase of acute infections, certain cells such

as those associated with the immune system and production of acute phase proteins have an increased anabolic response to the disease. Free aromatic amino acids of the plasma (phenylalanine and tyrosine) and sulfur-containing ones (methionine) are increased, while the concentration of branched-chain amino acids (isoleucine, leucine, and valine) are decreased during the early stages of acute infectious disease in man and experimental animals. The latter amino acids are apparently utilized at increased rates by skeletal muscle as a source of energy. Thus, feeding of a diet which is high in branched-chain amino acid as well as calories does reduce the protein wasting which is associated with infectious disease. Because of anorexia or malfunctioning intestinal tract, it may be necessary to give these nutrients parenterally.

Alterations in carbohydrates metabolism

In an acute infectious disease, the rates of glucose turnover and oxidation are generally elevated compared to a noninfected individual. The liver of an infected host has an increased propensity to produce glucose from gluconeogenic substrates. This can result in modest hyperglycemia in the early febrile phase of many infectious illnesses. In some infections associated with mild hepatic damage, glucose intolerance and insulin resistance can become quite severe, resulting in marked hyperglycemia and glucouria. If the infectious disease leads to hepatic failure, such as in severe gram-negative sepsis or viral hepatitis severe hypoglycemia can develop which may require an immediate infusion of glucose. Thus, the alterations in carbohydrate metabolism are complex and vary depending on the duration and variety of infectious disease. In general feeding of glucose will result in

some decrease in protein wasting associated with infectious disease, but it also tends to reduce the function and protein synthetic ability of cells involved in the host defense against infectious disease.

Alterations in lipid metabolism

In most infectious diseases, the host can utilize lipid calories as a source of energy but at a reduced efficiency compared to the noninfected individual. There is a slightly reduced ability to clear lipids from the blood and a decreased capacity of the liver of the infected host to synthesize ketones from long-chain fatty acids. This results in a reduced ability to develop starvation ketosis and increases the dependence on glucose calories as a source of energy. Thus, the infected host must break down body proteins to obtain amino acids to meet energy requirements and permit the increased synthesis of glucose.

Alterations in electrolyte, trace elements, and vitamin metabolism

With the onset of fever, there is a marked retention of sodium chloride by an infected individual. Plasma concentrations of iron, and to a lesser entent zinc, are markedly diminished during an acute infectious illness. This represents a redistribution and accumulation of these trace metals in the liver. In severe bacterial infections, iron may virtually disappear from the plasma, leading to anemia, if the infection becomes chronic. Increasing dietary intake of iron does not correct this response and may in fact be harmful to the host defense against the infectious disease. In contrast, plasma copper concentrations increase due to increased production of its carrier-protein (ceruloplasmin). The metabolism of most vitamins is not appreciably altered by infectious disease.

Nutrient requirements during infectious disease

Alterations in nutrient requirements depend on the severity and duration of the infectious disease. In severe infections protein requirements may be increased by 50%. Caloric needs are also increased during infectious disease and may increase to 200-300% of the resting requirements depending on the magnitude of the fever and duration of the illness. While the requirements for trace elements and vitamins do not appear to be elevated appreciably by the presence of acute infection, most experts recommend a two-fold increase in the intake of these nutrients. In a previously well-nourished individual, the losses of body nutrients during an acute infection of short duration are generally reconstituted during the first several weeks of convalescence. However, if the patient has a life-threatening infectious disease and has lost 10% or more of his body weight, nutrient support therapy via the parenteral or enteral routes should be initiated to prevent rapid deveolpment of protein-calorie malnutrition and suppression of immune function.

Selected reading

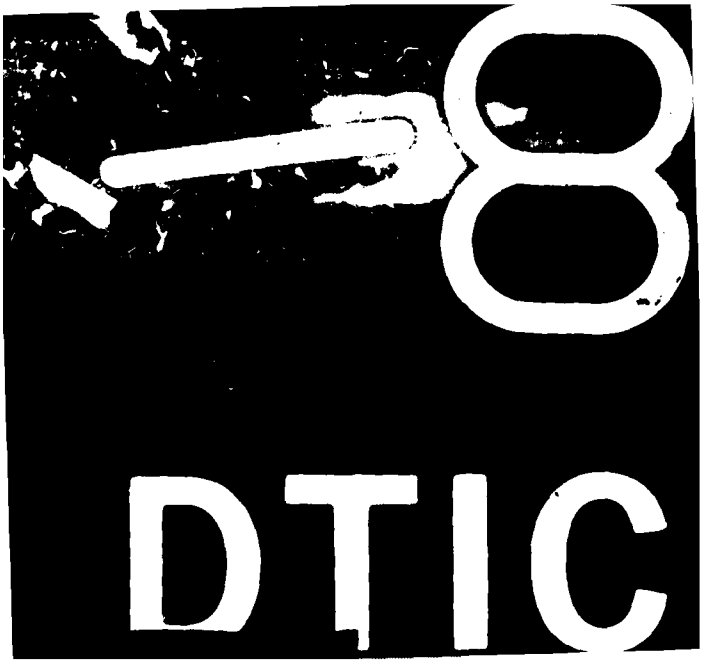
Beisel WR: Magnitude of host nutritional response to infection. Am J Clin Nutr 30:1236, 1977.

Beisel WR and Wannemacher RW Jr: Gluconeogenesis, ureagenesis, and ketogenesis during sepsis. JPEN 4:277, 1980.

Wannemacher RW Jr: Key role of various individual amino acids in host response to infection. Am J Clin Nutr 30:1269, 1977.

Wannemacher, RW Jr and Beisel WR: Metabolic response of the host to infectious disease. In "Nutritional Aspects of Care of the Critically Ill," JR Richards and JM Kinney (eds.), Churchill-Livingstone, Edinburgh, p. 135, 1977.

Wilmore DW and Kinney JM: Panel report on a nutritional support of patients with trauma or infection. Am J Clin Nutr 34:1213, 1981.


8
DTIC